

Thirdly, what would have constituted adequate supervision in this case? It might have begun with preparation for the school trip, for example by ensuring adequate medication to prevent seizures and making sure that the people in charge of the trip knew about triggering events or stressors and their management. Besag makes excellent suggestions about supervising the actual swimming activity. Perhaps some consideration should also be given to the routine wearing of a flotation vest in such cases, even though this might be considered restrictive. Unfortunately, there is little research in this subject, so it is difficult to design a package of protections with confidence.

Finally, what is the role of educating the individual with the seizure disorder, in this case a 14 year old child, about risks and prevention strategies? Smith believes that teaching water skills to children is an important measure in preventing drowning⁹ and cites the research of Asher et al¹⁰ as providing the first comprehensive evidence that teaching swimming and water skills may usefully supplement other strategies such as fencing pools. Barss, however, disagreed and argued that more definitive studies are needed to assess the benefit of such programmes.¹¹ No specific studies have been done among children with disabilities.

Though we need to assess and minimise risk, at the same time every effort should be made to integrate individuals with special healthcare needs into society. Thus swimming should not be discouraged in people with epilepsy or any other physical disability. But practical precautions can and ought to be taken to minimise their risk of injury or death until research

brings forth proved preventive strategies. Despite the dramatic nature of such cases, they are still rare.

Samuel N Forjuoh *associate professor and director of research*

Department of Family and Community Medicine, Scott and White Memorial Hospital and Foundation, Texas A&M University System HSC College of Medicine 1402 West Avenue H, Temple, TX 75604, USA

Bernard Guyer *professor and chair*

Department of Population and Family Health Sciences, Johns Hopkins School of Hygiene and Public Health, 615 N Wolfe Street, Baltimore, MD 21205, USA

- 1 Diekema DS, Quan L, Holt VL. Epilepsy as a risk factor for submersion injury in children. *Pediatrics* 1993;91:612-6.
- 2 Josty IC, Narayanan V, Dickson WA. Burns in patients with epilepsy: changes in epidemiology and implications for burn treatment and prevention. *Epilepsia* 2000;41:453-6.
- 3 Forjuoh SN, Guyer B, Strobino DM, Keyl PM, Diener-West M, Smith GS. Risk factors for childhood burns: a case-control study of Ghanaian children. *J Epidemiol Community Health* 1995;49:189-93.
- 4 Roberts I, Norton R. Sensory deficit and the risk of pedestrian injury. *Inj Prev* 1995;1:12-4.
- 5 Besag FMC. Tonic seizures are a particular risk factor for drowning in people with epilepsy. *BMJ* 2001;322:975-6.
- 6 National Committee for Injury Prevention and Control. *Injury prevention: meeting the challenge*. New York: Oxford University Press, 1989.
- 7 Long L, Reeves AL, Moore JL, Roach J, Pickering CT. An assessment of epilepsy patients' knowledge of their disorder. *Epilepsia* 2000;41:727-31.
- 8 Kankirawatana P. Epilepsy awareness among school teachers in Thailand. *Epilepsia* 1999;40:497-501.
- 9 Smith GS. Drowning prevention in children: the need for new strategies. *Inj Prev* 1995;1:216-7.
- 10 Asher KN, Rivara FP, Felix D, Vance L, Dunne R. Water safety training as a potential means of reducing risk of young children's drowning. *Inj Prev* 1995;1:228-33.
- 11 Barss P. Cautionary notes on teaching water safety skills. *Inj Prev* 1995;1:218-9.

Do we need specialist adolescent units in hospitals?

Possibly

Papers p 957

If you were an adolescent aged 12-19 and you needed to be admitted to hospital would you want to be admitted to a paediatric unit with young children, an adult ward, or a separate unit just for adolescents? And if the last was your choice, what arguments would you put forward to justify it? A paper in this week's issue puts forward several.

One argument for separate adolescent wards is that professionals skilled in the care of young people create a "therapeutic environment" that might be especially beneficial. However, this is difficult to prove and no one has undertaken a controlled study to identify such an impact. Most obviously it could be argued that properly organised adolescent units provide for the specific developmental needs of those in the second decade of life—schooling, recreation, socialising—as well as for their increased needs for privacy. Additionally, such facilities have the potential for enhanced medical services at the interface between medical, psychiatric, and substance misuse treatments. Further, for young adults with chronic diseases, who may be developmentally less mature than their peers, the adolescent unit provides a more ideal hospital environment than those provided by either child or adult wards.

Yet are these arguments enough to justify separate hospital facilities specifically set aside for adolescents?

Are the teenage years so unique? Or is the age group 12-19 years so heterogeneous—socially, physiologically, and emotionally—as to make such facilities inefficient, ineffective, and ill conceived? Are the issues that lead to the admission of adolescent males (predominantly trauma) and adolescent females (predominantly obstetric) sufficiently different to warrant substantially different clinical and emotional support systems?

Russell Viner, the foremost British expert on hospital adolescent medicine, cogently argues that there are enough adolescents being admitted to district general hospitals as inpatients or for day case treatment to warrant a dedicated adolescent hospital ward in most of these hospitals (p 957).¹ Others would concur. In their survey of hospitals in England and Wales, Suresh et al report that 26% of the 225 hospitals surveyed had made some accommodation for their adolescent patients.² Most had a separate bay for adolescents within a paediatric ward, but 16 hospitals (nine district and seven university hospitals) had separate units. Exactly what lead to the establishment of units where they exist is unclear, but the authors suggest that it was probably available funding coupled with interested clinicians.

The call for separate units for adolescents in hospitals in the United Kingdom is not new. In 1959 the Platt report acknowledged that "the requirements of adoles-

BMJ 2001;322:941-2

cents differed from those of adults and children and ideally adolescents need their own accommodation.”³ What is new is that Viner has worked out that enough adolescents are admitted to British hospitals to warrant making such accommodation available.

In the United States during the 1970s and 1980s there was a tremendous push to establish inpatient adolescent units. By the mid-1990s the Society for Adolescent Medicine, the leading US professional organisation for adolescent health, estimated that there were 40-60 such units in the United States. As in Britain, some of these units are simply sections within other wards. The Society for Adolescent Medicine continues, however, to advocate “the continuation and establishment of adolescent medicine inpatient units in both paediatric and general hospitals as an optimal approach to the delivery of developmentally appropriate health care to hospitalised adolescent.”³ If Viner is correct this ideal can and should be realised in many district hospitals in the United Kingdom.

But even where the numbers do not justify a separate ward for adolescents, a multidisciplinary approach from health professionals with interest and expertise in adolescent health is still feasible in every hospital. As

the Society for Adolescent Medicine suggests, this will be achieved through establishing guidelines for the managing teenagers in hospital, so that those with greatest expertise can be involved with young people's care.⁴ But to truly realise a vision where all young people can receive the comprehensive services they need to become healthy adults we need to ensure that all health professionals in both primary and secondary care have the training they need to provide optimal care for this age group.

Aidan Macfarlane *international freelance consultant in strategic planning of child and adolescent services*

Oxford OX1 4LJ (Aidanmacfa@aol.com)

Robert W Blum *professor and director*

Center for Adolescent Health, University of Minnesota, 200 Oak Street SE, Minneapolis, MN, 55455-2002

- 1 Viner R. National survey of use of hospital beds by adolescents aged 12 to 19 in the United Kingdom. *BMJ* 2001;322:957-8.
- 2 Suresh S, Doull IJM, Thomas P. Adolescent inpatient units. *Arch Dis Childhood* 2000;82:266.
- 3 Watson S. A ward of their own. *Nursing Standard* 1998;12:12.
- 4 Fisher M, Kaufman M. Adolescent inpatient units: a position state of the society for adolescent medicine. *J Adolescent Health* 1996;18:307-8.

Haematuria in asymptomatic individuals

It is often caused by inherited thinning of the glomerular membrane

Haematuria is often detected incidentally by “dipstick” tests in clinical practice, and much recent discussion in the *BMJ* has centred round whether haematuria in asymptomatic individuals should always be investigated or whether it can be disregarded.^{1,2} Certainly haematuria can sometimes be dismissed as due to contamination with menstrual blood or to a urinary tract infection, but in other cases, as one correspondent chided, why do the test if you are going to ignore the result?

In most cases of dipstick haematuria the next step should be to examine the urine by phase contrast microscopy to confirm the haematuria and determine whether the red cells have originated from the glomerulus or elsewhere in the urinary tract.³ “Dysmorphic” or “glomerular” red cells are present when there is glomerulonephritis with proliferative features and “non-glomerular” red cells when bleeding is from elsewhere in the urinary tract, usually resulting from infections, stones, a tumour, or contamination. Finding haematuria without proteinuria cannot be used to infer a non-glomerular origin since glomerular bleeding is not necessarily accompanied by proteinuria.³

What is the usual source of haematuria in asymptomatic individuals? The reported prevalence of haematuria in the community varies from <1% to 14%, but most studies of the causes of haematuria have examined patients referred to nephrology or urology clinics and have not used phase contrast microscopy to differentiate between glomerular and non-glomerular sources. In contrast, a recent Australian survey found that 9.4% of community based adults aged 25 or over had haematuria (SJ Chadban et al, scientific meeting of

the Australian and New Zealand Society of Nephrology, 2000), and two thirds of these had red cells originating from the glomerulus on phase contrast microscopy (SJ Chadban, personal communication). Thus haematuria in otherwise well adults is more often due to bleeding from the glomerulus than from elsewhere in the urinary tract.

What causes this glomerular bleeding? In unselected individuals with glomerular haematuria the renal biopsy most often shows thin basement membrane disease.⁴ This condition is characterised by uniform thinning of the glomerular basement membrane on ultrastructural examination and a very mild proliferative glomerulonephritis. Thin basement membrane disease is also known as benign familial haematuria,⁵ and other family members often have haematuria too. Affected individuals typically have lifelong glomerular haematuria, minimal proteinuria, and normal renal function as well as often having a family history.

Alport's syndrome is a better understood but less common inherited disease that also affects the glomerular basement membrane. In Alport's syndrome the membrane is lamellated rather than thinned, and affected individuals have haematuria, renal failure, and often deafness and ocular abnormalities. The inheritance is either X linked or autosomal recessive, when mutations occur in the COL4A5 and COL4A3/COL4A4 genes respectively. These code for the $\alpha 5$, $\alpha 3$, and $\alpha 4$ chains of type IV collagen, the major constituent of the glomerular basement membrane. The demonstration of thinned glomerular membranes in carriers of autosomal recessive Alport's syndrome first suggested that mutations in thin basement

BMJ 2001;322:942-3